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MASTOPHORA AND THE MASTOPHOREAE: GENUS AND SUBFAMILY OF CORALLINACEAE

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The genus *Mastophora* was proposed and properly validated by Decaisne early in 1842, but, because of his arrangement of topics, it has been generally misunderstood by later writers, especially as to identity of its type species. Later in 1842, Decaisne in a separate paper reduced his genus under *Melobesia* and this has assisted in the confusion. The two papers of Decaisne, (1) "*Essais sur une classification des algues*" (*Ann. Sci. Nat., bot.*, ser. 2, **17:** 297–380, pl. 14–17, 1842) and (2), "Mémoires sur les Corallines" (*Ann. Sci. Nat., bot.*, ser. 2, **18:** 96–128, 1842) were reprinted (and repaged) as "Thèses présentées et soutenues à la Faculté des Sciences de Paris, le 19 Decembre 1842," and were better known in this form to Harvey, Kuetzing and Areschoug, who shaped our knowledge of the Coralline Algae when these organisms were becoming recognized as plants rather than as being relegated to the animal kingdom. The inclusion of the two papers in one and with consecutive paging is partly responsible for misstatements occurring from 1842 onward.

On p. 69 of the reprint (p. 365 of vol. **17** of the *Ann. Sci. Nat., bot.*, ser. 2), Decaisne proposes *Mastophora* as a new genus, gives a short but distinctive Latin diagnosis, designates it as a marine alga of the habit of a *Lichen* or of a *Zonaria*, and names a type specimen: "*exsicc.* no. 2232" of "Cumming (or ⌈Cuming?⌋ from Manilla." On pp. 63 and 84 of his reprint (pp. 359 and 380 of vol. **17**, *Ann. Sci. Nat., bot.*, ser. 2, 1842) Decaisne gives an outline of the genera of his "Corallineae" and here as well as in his explanation of plate 17, fig. 11, he coins the binomial *Mastophora lichenoformis* Decaisne, giving a thoroughly satisfactory figure of a branch of the plant, together with (under Fig. 11*a*) a vertical section through a tetrasporic conceptacle showing the single apical pore and the supporting distinctly monostromatic thallus. In consequence, it may be emphasized that *Mastophora* was provided by Decaisne with a Latin diagnosis, a type

species with a type specimen (*Mastophora licheniformis* Dec'ne and Cuming's Exss. No. 2232) and distinctive illustrations.

In his second paper, "Mémoire sur les Corallines" (pp. 85–116 of the reprint and pp. 96–128 of vol. **18** of *Ann. Sci. Nat., bot.*, ser. 2, 1842), Decaisne says (p. 127, p. 115 of the reprint) that *Melobesia* varies considerably in consistency and form, and that he has changed his point of view as to the importance of different modes of division of the fronds as well as of completeness, or lack of it, of adherence to the substratum. He therefore, in consequence of more profound study, reduces his *Mastophora* to a section of *Melobesia*. He names first *Melobesia* (*Mastophora*) *licheniformis* Decaisne (with *Zonaria rosea* C. Ag., non Lamour. as a synonym), second *M. Lamourouxii* Dec'ne (with *Padina rosea* Lamour. herb., *Dictyota rosea* Lamour. and *Peysonnelia* Dec'ne Pl. Arab., as synonyms) and, as a third species, describes *Melobesia tenuis* Dec'ne from the Sandwich Islands. It is to be noted that the three binomials of this second deliverance of Decaisne are *Melobesia* (*Mastophora*) *licheniformis*, *Melobesia* (*Mastophora*) *Lamourouxii* and *Melobesia* (*Mastophora*) *tenuis*, all new species of Decaisne, but practically never referred to under their proper binomials in all the succeeding phycological literature. They are, partially on this account, much confused. On p. 127 (p. 115 of the reprint) Decaisne also makes it clear that he does *not* consider *Melobesia* (*Mastophora*) *licheniformis*, his type of this genus *Mastophora*, to be synonymous with the *Millepora lichenoides* Ellis et Solander, but that the latter is very similar to his *Melobesia* (sectio I, *Melobesiae verae*) *verrucata* Lamour. Practically every phycologist who has dealt with these matters, from Kuetzing (1849) and Areschoug (1852) to De Toni (1905), has placed the "*Mastophora licheniformis*" Dec'ne under *Lithothamnium lichenoides* (Ell. et Sol.) Heydrich, misled apparently by the similarity of names, and without careful consideration of the complete text of Decaisne.

The genus *Mastophora* Decaisne, fully validated and with its type species, *M. licheniformis* Decaisne, fully illustrated, and with type specimen No. 2232 Cuming from the type locality Manila definitely indicated, has been recognized by all later writers as a proper genus, but the species he referred to it have been very much confused. Their distribution is tropical and subtropical Indo-Pacific. It is impossible to determine with exactness what species are to be recognized and what names are to be applied without a careful study of the types, and this is at present impossible. The following notes, however, may indicate the present complexity of the problem.

The genus *Mastophora* as defined and illustrated by Decaisne seems to be a genus limited to one, or possibly to two, species. It is definitely monostromatic, its layers are not normally superposed, its calcification is strong, so it is not flexible, its branching (or lobing) is lateral and pin-

nate, it is more or less decumbent, and its attachment is through adventitious, elongated unicellular rhizoids with more or less expanded tips. It does not possess a distinct thickened stipe or midribs. Its conceptacles are large, mammiform and strongly projecting, opening in all cases, by a single pore.

Mastophora Decaisne (1842)

Decaisne, *Ann. Sci. Nat., bot.*, ser. 2, **17:** 359, 1842; *Melobesia* Sect. III *Mastophora* Decaisne, *Ann. Sci. Nat., bot.*, ser. 2, **18:** 126, 1842. *Mastophora* Auctt. recentior., p.p.; *Phlyctidium* Montagne (MS.?), *Voy. au Pole Sud.*, 148, 1845.

Mastophora rosea (C. Ag.) comb. nov.

M(astophora) rosea Rosanoff, *Mem. Soc. imp. soc. nat. Cherbourg*, **12:** 13, 1866 (?) (nomen!); *Zonaria rosea* C. A. Agardh, *Syst. Alg.*, 264, 1824; Gaudichaud *in* Freycinet, *Voyage Uranie et le Physicienne, bot.*, 164, 1826. (The names *Padina rosea* Paliser de Beauvois, Ms., and *Dictyota rosea* Lamouroux, *Nouv. Bull. Soc. Philom., Paris*, **1:** 33, 1809, while possibly applying to this species or not, were never validated.) C. A. Agardh gives a proper diagnosis. Type locality Guam, in the Marianas (or Ladrones) Archipelago; *Mastophora licheniformis* Decaisne, *Ann. Sci. Nat., bot.*, ser. 2, **17:** 359 (p. 63 of reprint) (binomial), 365 (p. 69 of reprint) (genus and type specimen), 380 (p. 84 of reprint), binomial and descr. figures, pl. 17, figs. 11, 11*a*, 1842 type locality Manila, P. I.; *Melobesia* (*Mastophora*) *licheniformis* Decaisne, *Ann. Sci. Nat., bot.*, ser. 2, **18:** 126 (p. 114 of reprint), 1842; *Mastophora macrocarpa* Montagne, *Voy. au Pol. Sud*, 149, 1847 type locality Guam, in Marianas (or Ladrones) Arch.; Kuetzing, *Spec. Alg.*, 697, 1849, *Tab. Phyc.*, **8:** 48, pl. 100, I, 1858; Areschoug *in* J. Ag., *Spec. Alg.*, 2: 2: 528, 1852; De Toni, *Syll. Alg.*, 4, 4: 1776, 1905, *Ibid.*, 6, **5:** 695, 1924; Heydrich, *Hedwigia*, **33:** 300, 1894; Weber von Bosse et Foslie, *Siboga Exp., Mon.* **61:** 70, 71, txt. fig. 27, pl. 13, 1904; Setchell, *Univ. Calif. Pub. Bot.*, **12:** 109, 1926; *Melobesia foliacea* Kuetz., *Phycol. gen.*, 386, 1843, *Spec. Alg.*, 696, 1849; *Mastophora foliacea* Kuetz., *Tab. Phyc.*, **8:** 48, pl. 100, II, 1858 (type locality, Marianas (or Ladrones Arch., Sonder, *Die Algen von trop. Austral. Abh. Gebiete Naturwissench. Ver. zu Hamburg*, **5** (2): 54, 1871 (locality Cape York); *Mastophora plana* De Toni, *Syll. Alg.*, 4, 4: 1775, 1905 (*non M. plana* Sonder); *Mastophora lichenoides* Auctt. var. p.p.

Mastophora rosea f. condensata (Foslie) comb. nov.

Mastophora macrocarpa forma, Foslie, *Siboga Exp., Mon.* **61:** 71, txt. fig. 27, 1904; *Mastophora macrocarpa* f. *condensata* Foslie, *Algol. Notis.*, **IV:** 30, 1907; De Toni, *Syll. Alg.*, 6, 5: 695, 1924. *Mastophora pygmaea* Heydrich, *Hedwigia*, **33:** 300, pl. 15, fig. 16, 1894?.

Mastophora tenuis (Decaisne) Aresch.

Areschoug *in* J. Ag., *Spec. Alg.*, 2, 2: 528, 1852; Kuetzing, *Spec. Alg.*, 697, 1849; De Toni, *Syll. Alg.*, 4, 4: 1777, 1905; *Melobesia* (*Mastophora*) *tenuis* Decaisne, *Ann. Sci. Nat., bot.*, ser. 2, **18:** 127 (p. 115 of reprint), 1842, type locality, Hawaiian Islands. This may prove to be a form of the preceding. Specimens from Waikiki, Oahu, Hawaiian Islands, seem referable to *M. rosea.*

Mastophora affinis Foslie

Foslie, *Siboga Exp., Mon.* **61:** 71–73, txt. figs. 28, 29, 1904. Type locality, Sikka, Island of Flores, Lesser Sunda Islands. *Mastophora macrocarpa* f. *affinis* Foslie, *Algol. Notis.*, **VI:** 53, 1909; Foslie (ed. Printz), *Contrib. Mon. Litholh.*, 47, pl. 74, figs. 7–9, 1929.

This species, together with *M. pacifica* (Heydrich) Foslie from Formosa, seems possibly to be nearer to *Lithoporella* Foslie, but Foslie refers it finally (1909) under *M. macrocarpa.*

Metamastophora gen. nov.

Frons erecta aut adscendens, libera, flexilis, tenuiter calcarea, basi per rhizoides affixa, dichotome aut flabellate ramosa, primo monostromatica mox oligo- usque ad (in stipitibus costisque) polystromatica, cellulis textorum parve differentiatis, in superficiebus ut seriebus successive curvatis apparentibus; rhizoidibus basalibus conceptaculis omnibus magnis, e superficiebus bifariante et impariter prominentibus, poro singulo apertis; tetrasporangiis 4 partitis, conceptaculi fundum totum vestientibus; gonimoblastis peripheric is? Genus australe, subtropicum.

Species typica: *Melobesia* (*Mastophora*) *flabellata* Sonder, Australia, S. W.

It seems best to refer the very complex species from South Australia and South Africa placed under the genus *Mastophora* by writers later than Decaisne and which differ fundamentally from the simple *Mastophora rosea*, to a new genus, for which the generic name *Metamastophora* is proposed. Very little study has been made of these species and material of them is infrequent in collections. Much more study is needed of their vegetative structure, of their reproductive organs, and of the development of every kind of conceptacle. Of the probable 5 species, material of only one is accessible. The following outline, therefore, must be regarded as tentative.

A. Branching divaricate, terminal segments broadly cuneate, interruptedly revolute on apex and margins.

1. **Metamastophora flabellata** (Sonder) comb. nov.

Melobesia (*Mastophora*) *flabellata* O. G. Sonder, *Bot. Zeit.*, 1845, 55, type locality, mouth of Swan River, S. W. Australia; O. G. Sonder *in* Lehmann's *Plantae Preissianae*, fasc. **2:** 188, 1847; *Mastophora flabellata* Harvey, *Ner. Austr.*, 108, 1849; Kuetzing, *Spec. Alg.*, 697, 1849, *Tab. Phyc.*, **8:** 47, pl. 97, 1858; *Mastophora Lamourouxii* (p.p.), Aresch. *in* J. Ag., *Spec. Alg.* 2, 2: 526, 1852; Harvey, *Phyc. Austral.*, **5:** p. xxx, 1863; De Toni *Syll. Alg.* 4, 4: 1774, 1905, *Ibid.*, 6, 5: 695, 1924; Foslie (ed. Printz), *Contrib. Mon. Lithoth.*, **47** (f. *typica*), pl. 75, figs. 1–3, 1929. S. W. to S. E. Australia.

The Sonder species seems to be an independent entity, but it has commonly been referred under the binomial *Mastophora Lamourouxii*, which will be discussed later on. It may be mentioned here, however, that this binomial is to be considered a *nomen nudum* in the strictest sense. In contrast to the slender South African species referred to Decaisne's second species (Melobesia tenuis), the Australian plants of Sonder are relatively firmer, of more than one layer of cells (except at the very tips) and are divaricately flabellate in the lobing of their broadly cuneiform lobes. The plate of Kuetzing (*Tab. Phyc.*, **8**, pl. 97, 1858) shows these characters plainly and they are evident in a series of specimens at my disposal in Herb. Univ. Calif. The under surfaces of the segments seem only slightly pruinose.

2. **Metamastophora Lamourouxii** (Dec'ne ex Harvey) comb. nov.

Melobesia (*Mastophora*) *Lamourouxii* Decaisne, *Ann. Sci. Nat., bot.*, ser. 2, **18:** 126 (p. 114 of reprint), 1842 (?), nom. nud.!, *Mastophora Lamourouxii* Endlicher, *Gen. Pl., supp.* **3:** 50, 1843 (nomen!); Krauss, *Flora*, 1846, 211 (nomen!); Harvey, *Ner. Austr.*, 108, pl. 41, 1849 (descr. and illustration); Kuetzing, *Spec. Alg.*, 697, 1849 (sub. *Dictyota rosea* Lamour.), *Tab. Phyc.*, **8:** 47, pl. 98, II, 1858; Areschoug *in* J. Ag., *Spec. Alg.*, 2, 2: 526, 1852 (p.p.); De Toni, *Syll. Alg.*, 4, 4: 1774, 1905 (p.p.), *Ibid.*, 6, **5:** 695, 1924 (p.p.); Foslie (ed. Printz) *Contrib. Mon. Lithoth.*, 47, excl. plates and references.

The application of the name "*Lamourouxii*" to any *Mastophora* or *Metamastophora* is attended with the utmost of uncertainty. Decaisne does not describe his *Melobesia* (*Mastophora*) *Lamourouxii*, but he does refer to it the *Padina rosea* of Lamouroux *in herb.*, the *Dictyota rosea* Lamouroux, presumably of 1809, but not described, and an indefinite reference to *Peyssonnelia?* of "Dec'ne, Pl. Arab." He definitely separates it from the *Zonaria rosea* C. Ag. (not Lamour.) and seems to indicate that it may occur in the Red Sea, as Kuetzing has suggested. How the name came to be associated with the Port Natal plant, as has been done by Krauss and Harvey, does not appear. Harvey, who seems responsible

for this association, does not indicate as he often does the source of his information. He states only that he has seen dried specimens in the herbarium of Trinity College, Dublin, without indicating whether they were other than those of Gueinzius from Port Natal. One may doubt, therefore, the propriety of quoting Decaisne in connection with this species, unless one has the opportunity of seeing his type specimen.

Whether *Metamastophora Lamourouxii*, of S. E. Africa, obtains the right to its specific name from Decaisne or from Harvey, it certainly appears from the literature to be a distinct species. It seems to differ from *M. flabellata* of Australia by the thinner alae (monostromatic), the lack of divaricate lobing at the extremities of the somewhat less pronounced cuneate blades, and the under surfaces more pruinose. Kuetzing (loc. cit., 1858) certainly represents the alae of this species as monostromatic, while he represents the alae of *M. flabellata* as chiefly di-oligo-stromatic as is indicated by our specimens.

B. Branches oblique, strict; terminal segments not strictly cuneate, entire or lobed at apex, revolute along margins.

3. **Metamastophora canaliculata** (Harv.) comb. nov.

Mastophora canaliculata Harvey *in* Hook. f., *Flora Tasmanica*, **2:** 310, Feb. 15, 1859, *Alg. Austral. Exsicc.*, No. 443, *Phycol. Austral.*, 5, XXXI, pl. 263, 1863; Rosanoff, *Mem. Soc. Imp. Sci. Nat.*, **12:** 13, 1866; De Toni, *Syll. Alg.*, 4, 4: 1776, 1905, *Ibid.* 6, **5:** 695, 1924; Foslie (ed. Printz), *Contrib. Mon. Litholh.*, 47, pl. 73, figs. 10–12, 1929.

From the figures of Harvey (1863) and of Foslie (1929), the latter presumably from Harvey's distribution, this species is very distinct in habit and texture, probably approaching the next species in these respects. The dichotomies are obliquely erect, involute or canaliculate throughout, and glabrous and concolorous on both surfaces. Judging from figure 4 of Harvey's plate 263, the alae are polystromatic. This is seemingly a very distinct species, the type specimens from Tasmania, others from neighboring S. E. Australia.

4. **Metamastophora stelligera** (Endlicher et Diesing) comb. nov.

Mastophora stelligera Endlicher et Diesing, *Bot. Zeit.*, 1845, 290 type from Port Natal, South Africa; Kuetzing, *Spec. Alg.*, 697, 1849; Areschoug *in* J. Ag., *Spec. Alg.*, 2, 2: 528, 1852; De Toni, *Syll. Alg.*, 4, 4: 1777, 1905, *Ibid.*, 6, 5: 695, 1924. *Mastophora hypoleuca* Harvey, *Nereis Austr.*, 108, pl. 41, figs. 1–3, 1849, type locality, Port Natal, South Africa, leg. Dr. Guienzius; Areschoug *in* J. Ag., *Spec. Alg.*, 2, 2: 527, 1852; De Toni, *Syll. Alg.*, 4, 4: 1775, 1905; Foslie (ed. Printz), *Contrib. Mon. Litholh.*, 47, pl. 75, fig. 1, 1929.

While it has been impossible to compare the type of the species of Endlicher and Diesing with that of Harvey's species, or, in fact, to examine any specimen at all, it seems proper to consider the two species, identical in type locality and description, as most probably being one and the same. The plants are described as elate, with stipes and branches as narrowly furcate and canaliculate, the terminal blades as lanceolate, with blunt entire or bifid apices, and with margins revolute. The under surfaces of the blades are white-farinose or white-lanate, with depressed dark-colored spots. The conceptacles are described as being uniseriate along both margins of the blades. The upper surface of the blade is dull brownish purple and, according to Harvey, "shagrined and striate." It seems to be a complex and very striking species if the lower surface is correctly described and the further study of it is most desirable.

C. Branching more or less divaricate; frond flat throughout, and with plane margins; lobes blunt, entire.

5. **Metamastophora plana** (Sonder) comb. nov.

Melobesia (*Mastophora*) *plana* O. G. Sonder, *Bot. Zeit.*, 1845, 55, type locality, mouth of Swan River, S. W. Australia, leg. Preiss.; O. G. Sonder *in* Lehmann, *Pl. Preiss.*, **2:** 188, 1847; *Mastophora plana* Harvey, *Nereis Austr.*, 108, 1849; Kuetzing, *Spec. Alg.*, 697, 1849, *Tab. Phyc.*, **8:** 47, pl. 98, I, 1858; Harvey, *Phyc. Austral.*, **5:** xxx, 1863; Schmitz und Hauptfleisch *in* Engler und Prantl, *Die Natürl. Pfl. fam.*, I, **2:** 540, fig. 286*c*, 542, 1897; De Toni, *Syll. Alg.*, 4, 4: 1775, 1905 (p.p.), *Ibid.*, 6, 5: 695, 1924 (p.p.); *Mastophora Lamourouxii* f. *plana* Foslie, *Algol. Notis.*, **V:** 18, 1908, Foslie (ed. Printz), *Contrib. Mon. Lithoth.*, 47, pl. 75, figs. 4, 5, 1929.

From the descriptions and figures, since no specimens are available, it seems fairly certain that this is a distinct species within the genus. The plane frond is very different from that described for any other species and the figures of Kuetzing, while incomplete as to structure, indicate a high degree of complexity.

The genus *Mastophora*, as founded by Decaisne, in his *Essais*, may be restricted as has been indicated earlier, to species of the simple structure of *M. rosea* (C. Ag.) Setchell. As extended by Endlicher and Diesing (1845), Sonder (1845), Harvey (1849), Kuetzing (1849) and Areschoug (1852), it becomes heterogeneous. Kuetzing, in 1849, included not only the more complex species included here, but also certain species such as: *Mastophora crassiuscula* (Kuetz.) Kuetz. (*Spec. Alg.*, 699, 1849), *M. laevis* (Kuetz.) Kuetz. (*Ibid.*, 699; *Lithophyllum laeve* Kuetz., *Bot. Zeit.*, **5:** 33, 1847, *non Lithophyllum laeve* Stroenfelt, *Algveg. Isl.*, 21, pl. 1, figs. 11, 12, 1886, a later homonym); *M. lichenoides* (Ell. et Sol.) Kuetz. (not

M. licheniformis Dec'ne), and *M. patena* (Harv.) Kuetz., as a first group and, as a second group, the several species later described by Foslie and referred to *Lithoporella* and *Litholepis*. The members of the second group are to be considered properly referred as will be shown later. The members of the first group are mostly of a definite life form and hemiparasitic. In another paper they will be referred to a new genus. Very puzzling is the *Mastophora* (*Lithostrata*) *lapidea* Foslie (*Algol. Notis.*, **II:** 27, 1906) which was later referred to *Lithoporella* (*Lithostrata*) *lapidea* Foslie (System. Bemerk., in *Det. Kongl. Norske Vidensk. Selskabs Skrifter*, 1909 (2): 59, 1909) and finally as "*Mastocarpus lapidea* Harv." Foslie(*in Contrib. Mon. Lithoth.* (ed. Printz), p. 47, pl. 73, figs. 5–7, 1929). No description of the fruiting organs is given nor any reference to the genus *Lithostrata* Heydrich (1905) of the *Squamariaceae*. The only suggestion that may be made is that, without examination of the type, it possibly may represent a species of Foslie's first proposal (1898) of *Goniolithon* (subgen. *Eugoniolithon*) and that it may be related to *G. papillosum* (Zan.) Foslie.

In "1902" (or more probably in 1903) Foslie (in Aarsberetning for "1902" in *Det. Kongl. Norske Vidensk. Selskabs Skrifter*) outlined some 8 groups, into which he proposed to divide the *Corallinaceae*. In that outline he proposed the "Mastophoreae" to include the genus *Mastophora* (in an extended sense). He gave no characters for his groups but only enumerated the genera to be included under each. He named special groups for each of the definitely parasitic genera: *Schmitziella*, *Chaetolithon* and *Choreonema*. He also included, but doubtfully, "*Hildenbrandia*."

The other 3 groups proposed by Foslie are *Lithothamnioneae*, *Melobesieae* and *Corallineae*. Disregarding the parasitic (or hemiparasitic) nature of the genera as group characteristics, and reviving some names in accordance with the "Rules of Nomenclature," there may be proposed a series of 5 groups, or subfamilies, as follows:

1. Frond crustiform to fruticulose, inarticulate, continuous (reduced in parasitic species)....2
1. Thallus articulate or jointed....5. *Corallineae*
2. Thallus monostromatic to partially polystromatic, but without differentiation of tissues....4. *Mastophoreae*
2. Thallus di-polystromatic, with differentiated tissues....3
3. Tetrasporangia in sori....1. *Sporolitheae*
3. Tetrasporangia in conceptacles....4
4. Tetrasporangial conceptacles opening by few to many pores....2. *Lithothamnieae*
4. Tetrasporangial conceptacles opening by a single pore....3. *Lithophylleae*

The *Mastophoreae*, differing from all other *Corallinaceae* by their lack of, or, at least, by their very feeble differentiation of tissues, include 5 genera, arranged as follows:

1. Fronds decumbent or ascending, monostromatic, brittle.....2
1. Fronds elate, becoming homogeneo—polystromatic, with stipe branching and terminal blades flexible.....5. *Metamastophora*
2. Fronds entire on margins, proliferating obliquely and lamellately, producing superposed layers devoid of rhizoids.....3
2. Fronds lobed on margins, neither proliferating nor superposed, attached by adventitious rhizoids.....4. *Mastophora*
3. Fronds superposed, agglutinated into a common crust.....3. *Goniolithon*
3. Fronds superposed, free.....4
4. Thallus cells palisade-like.....2. *Lithoporella*
4. Thallus cells nearly isodiametric.....1. *Litholepis*

THE PEDIGREED CULTURE OF PARAMECIUM AURELIA AT YALE UNIVERSITY

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It seems desirable, as a matter of record, to bring up to date the history of the long-continued pedigreed culture of Paramecium at Yale University, because it is now more than two decades since a summary appeared in these PROCEEDINGS.[1]

The culture was started on May 1, 1907, by the isolation of a "wild" specimen of *Paramecium aurelia* on a depression slide in about five drops of culture fluid. Descendants of this individual constitute the various lines of the pedigreed race. During the first eight months the culture medium consisted solely of hay infusion, but thereafter infusions of various materials common in the usual environment of Paramecia proved to be more favorable and were employed. The media have always been thoroughly boiled to prevent any possible contamination with foreign strains of Paramecium. In brief, the cells of the culture today are direct lineal descendants by division from the single animal isolated in 1907.

The main object of starting the culture was to determine whether Paramecium can reproduce by division indefinitely without recourse to conjugation. Therefore during the first eight years of the culture's life, to May 1, 1915, during which 5071 generations were attained, the possibility of conjugation was absolutely precluded by the daily observation and isolation of the products of division. After this date daily isolation was judged unnecessary, because the continued life and health of the culture had long since justified the conclusion that conjugation is not, as had been previously generally maintained, a *sine qua non*, under favorable environmen-

tal conditions, for the continued life of Paramecium and, presumably, of unicellular animals in general.

However, at the formal termination of the experiment in 1915, the culture was still maintained but without the exacting daily observation and recording of the division rate previously required. So, from this point, there is the possibility that conjugation between closely related individuals of the pedigreed culture may have occurred without being detected.

In this manner the culture has been maintained up to the present (March 1943). From time to time, thirty-day tests have been made of the division rate of the animals under the former rigid culture conditions, and in nearly every case the same general average division rate has been revealed as during the first eight years of life; that is, between 50 and 60 generations per month. There is no evidence of waning vitality. On the basis of these tests it is fair to estimate 600 generations attained each year since May 1, 1915, which gives, in round numbers, 21,800 generations attained by the culture during the nearly 36 years of its life to date. The vitality of the culture is further attested by the fact that it is continuously affording animals for various other experiments in the Osborn Zoölogical Laboratory and elsewhere.

It may be recalled that studies on this culture by Woodruff and Erdmann[2] revealed a periodic internal nuclear reorganization process to which the name endomixis was given. The establishment of endomixis raised new problems though obviously without affecting the basic conclusion that conjugation is not intrinsically a necessary phenomenon in the life history of the organism. If, however, it prove true that synkaryon formation may occur during reorganization in this race, as first stated by Diller,[3] so that the process becomes autogamic, then, of course, *self*-fertilization has not been excluded in the experiments.[4]

[1] Woodruff, L. L., "The Present Status of the Long-Continued Pedigreed Culture of *Paramecium aurelia* at Yale University," *Proc. Nat. Acad. Sci.*, **7**, 41–44 (1921).

[2] Woodruff, L. L., and Erdmann, R., "A Normal Periodic Reorganization Process without Cell Fusion in Paramecium," *Jour. Exp. Zoöl.*, **17**, 425–518 (1914).

[3] Diller, W. F., "Nuclear Reorganization Processes in *Paramecium aurelia*, with Descriptions of Autogamy and Hemixis," *Jour. Morph.*, **59**, 11–67 (1936).

[4] Woodruff, L. L., "Endomixis," *Protozoa in Biological Research*, edited by G. N. Calkins and F. M. Summers, Columbia University Press, 1941, pp. 646–665.

HIGH MUTATION FREQUENCY IN DROSOPHILA PSEUDOÖBSCURA, RACE B

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Variations in the mutation rate may be due to a number of factors in the external or internal environment of the organism. We can control many of these factors; others are unknown and appear spontaneously; they may persist for some time and then disappear again. In some cases this may be due to a selection of proper genic modifiers of the mutation rate. Modifying genes have been made responsible for the control of the mutation rate, since the mutation rate can be altered by hybridization; such a case has been described for *Drosophila pseudoöbscura*.[1] Several investigators have observed cases of high mutation frequencies in *D. melanogaster*[2, 3, 4]; other cases of the same nature are more doubtful. *D. pseudoöbscura*, race B, has furnished another example exhibiting the sudden rise of mutation frequency, to be recorded in this paper.

The phenomenon first appeared in the progeny of a pair mating, where four sex-linked mutants were observed. On outcrossing flies from this culture, mutations occurred again to an unexpected degree. The number of mutations per pair mating is quite variable. Some of the most striking cultures are recorded in table 1. In each case we deal with the progeny of only one pair mating. The average yield per culture is 242 flies; this number is based on counts of the progenies of 758 pair matings chosen at random; even in the largest progenies there are less than 500 flies per bottle.

TABLE 1

CULTURE NO.	TYPE OF MUTATION	NO. OF MUTATIONS
Original	*bd, y, ri,* (spread wings)	4
1045	*v, Aw, L,* (Abnormal)	4
1176	bd^{ci}, *dx, dy(m), Sc*	4
1671	*dy(m), L, Vg,* (Abnormal)	4
1691	*bd, sct, tt, Cy,* (Wing-mosaic)	5
1697	*bd, sct, com, asc, Sc,* (rough eye)	6
2654	*bd, dy(m), bbl, Aw,* (rough eye)	5
2676	*sct, w, sn(f), Aw, tt*	5
2708	*dy(m), tt, Tho,* (Rough eye)	4
2936	*bd, sct, Vg,* (Rough eye)	4
2971	*bd, com,* H^4, *Sb, Vg*	5
a177	*dy(m), com, asc, jv, dow,* (rough eye)	6

The mutations evidently may occur at any time during the development of the germ line or of the somatic line. Mutated individuals of a certain type may occur in numbers varying from one to fifty per cent of the offspring of a pair mating; in somatic mutations patches of varying sizes have been observed. There seems to be no preference as to the time of mutation. The numbers of mutations recorded in this paper refer to separate occurrences of mutation, not to numbers of individuals showing new mutant characters.

In our study only "visible" sex-linked recessives are used as indicators of the high mutation rate, because they are detected in the immediate offspring of a female with the abnormal rate. Autosomal recessives should not be used, because the probability of their detection depends on the mating system used, and even then it is not certain at what time and in which individual they have arisen. Dominants are disregarded to avoid confusion with another mechanism which is responsible for inducing mutations of the defiency type; we shall refer to this later. Although the high mutation rate is observed in males also, and although dominants and autosomal recessives are produced by the present mechanism, it is for the above reasons that we restrict our observations to visible sex-linked recessives arising in females.

The most likely cause of a phenomenon of this nature is a genic one. It has proved difficult, however, to demonstrate that a gene is responsible for the increase in the mutation rate. We deal here with a quantitative difference between the normal and the mutating strains; therefore, large numbers are required to make the data meaningful.

The normal spontaneous mutation rate for visible sex-linked recessives in *D. pseudoöbscura*, race B, is 0.01%. This number is based on the occurrence of four sex-linked recessives in 46,804 flies, from cultures preceding the time of the first observation of the high mutation rate, plus those from the pure Seattle strain.

In determining the abnormal mutation rate we shall consider only those cultures which have given at least one sex-linked recessive, since this is the only indication of the presence of the "Mutator" gene in the mother. Thus, we obtain a mutation rate per culture showing mutations, rather than a mutation rate per total number of flies. The higher the total mutation rate, the greater will be the coincidence of several sex-linked recessives in one culture, remembering that we always deal with the offspring of one female per culture and that the average number of offspring is the same for wild type females and Mutator females. With a normal mutation rate of 0.01% for sex-linked recessives it is obvious that two mutations would occur by chance in the same culture so rarely that the mutation rate per culture showing mutations is practically 1.00. Thus, from Seattle females seven mutations were obtained in seven cultures (rate = 1.00); from Morro fe-

males five mutations were obtained in five cultures (rate = 1.00). But from selected females 160 mutations were obtained in 102 cultures (rate = 1.57). The coincidence of mutations is directly related to the total mutation rate, so that by knowing one we can determine the other. Due to the small numbers, which are always a disadvantage in a study of this sort, the errors may be considerable; the calculations are also based on the assumption of a constant number of flies per bottle, which further decreases the accuracy of the result. Therefore, the rates which will be presented must be taken as approximations; but we are interested in getting some idea of the mutation frequency in flies heterozygous or homozygous for the Mutator gene. In general, the estimates are probably conservative, since the normal spontaneous mutation rate is included in the abnormal rate, which would tend to lower the coincidence.

Females resulting from a mating of a selected fly to wild type, regardless of the direction in which the cross is made, give a rate of 1.47 (100 mutations in 68 bottles giving mutations). This is the rate obtained from flies heterozygous for the Mutator gene. Assuming a Poisson distribution, that is, a population homogeneous for a factor increasing the mutation rate, a result like this would be obtained, if the rate of mutations per total number of flies were 0.34%. Thus, the normal spontaneous mutation process is increased thirty-four times with one dose of the Mutator gene.

If flies can be homozygous for the Mutator gene, we might expect a higher mutation rate from females that have resulted from a mating of both selected male and female; or, conversely, only if flies resulting from such a mating give more mutations, can we know that homozygous flies are produced and that they have a higher mutation rate than the heterozygous flies. Females resulting from such a mating give a rate of 1.76 (60 mutations in 34 cultures giving mutations). That is the rate obtained from females both of whose parents were probably heterozygous for the Mutator gene. From that mating we should expect one-fourth of the offspring to be homozygous for the Mutator gene one-half heterozygous and one-fourth wild type.

Let m be the expected mean frequency of occurrence of a mutation in the population of subsamples. Since m is known to be equal to 0.0034×242 for cultures resulting from heterozygous females, and equal to 0.0001×242 for cultures resulting from homozygous wild type females, the relative frequencies of subsamples with one mutation, two mutations, etc., are known for these two groups. Let k be the number of cultures resulting from homozygous Mutator females; then there are $k \frac{m}{e^m}$ cultures with one mutation in this group, $2k(0.3615)$ cultures with one mutation when the mother was heterozygous and $k(0.0234)$ cultures with one mutation when the mother was wild type; similarly for cultures with two, three, etc.,

mutations. We equate the theoretical total of cultures with one mutation to the observed number with one mutation, and do likewise for the cultures with two, three, etc., mutations; we obtain the following six equations:

$$k\frac{m}{e^m} + 2k(0.3615) + k(0.0234) = 21$$

$$k\left(\frac{m^2}{2e^m} + 0.2979\right) = 7$$

$$k\left(\frac{m^3}{6e^m} + 0.0960\right) = 2$$

$$k\left(\frac{m^4}{24e^m} + 0.0168\right) = 2$$

$$k\left(\frac{m^5}{120e^m} + 0.0028\right) = 1$$

$$k\left(\frac{m^6}{720e^m} + 0.0004\right) = 1$$

The number of mutations in the homozygous group is equal to km, in the heterozygous group it is equal to $2k(242)$ (0.0034), and in the wild type group it is equal to $k(242)$ (0.0001); the total number of mutations obtained was 60; therefore, we find $km + 2k(242)$ (0.0034) $+ k(242)$ (0.0001) = 60, or, $k = \frac{60}{1.67 + m}$. We can now determine the most probable value of m in the usual manner.[5] We find $m = 1.703$. Thus, the mutation rate of females homozygous for the Mutator is 0.70%, approximately twice that of the heterozygotes. This is a linear increase with the dosage. In this connection, it is interesting that the gene dotted in maize produces an exponential increase with the dosage as concerns mutations of the "a" gene.[6]

The offspring of heterozygous flies should have a total mutation rate of 0.17% if the above calculations are correct, since now half the flies are heterozygous and half are wild type; therefore the mutation rate should be one half of 0.34%. We actually obtain 7 mutations in 4679 flies which is a rate of 0.15%; this is in good agreement with the expectation.

The crosses designed to locate the Mutator gene are shown in table 2. All the females shown in the table should be heterozygous for the Mutator gene except those with the wild type chromosome whose homologue is responsible for carrying the Mutator gene; from these females we should expect a rate of 1.00 whereas all the others should give an average of 1.47. Thus, chromosome II would seem to be the carrier of the Mutator gene.

This, however, must be taken cum grano salis; not only are the numbers uncomfortably small for this kind of a test, but the dot chromosome which cannot be marked was not controlled. The conclusion, therefore, is tentative.

TABLE 2

Selected Stubble (II) or Scute (III) or Curly (IV) × Morro or Seattle

↓

Stubble or Scute or Curly / Morro or Seattle ♂♂ × Morro or Seattle ♀♀

↓ ♀♀

	NO. OF MUTATIONS	CULTURES SHOWING MUTATIONS	RATE
Selected II	23	14	1.64
Wild type II	4	4	1.00
Selected III	12	9	1.33
Wild type III	6	4	1.50
Selected IV	15	8	1.86
Wild Type IV	15	11	1.36

Table 3 shows the maps of the chromosomes; the map of the *X*-chromosome is based partially on the one published previously.[7] Not all of the mutants shown on the maps can be assumed to have been induced by the Mutator gene here under discussion; there is another mechanism at work in the same cultures which induces an extraordinarily high number of mutations of the deficiency type, mainly Smoky, Notch and Minutes. Sometimes it is difficult to tell the two apart, because in some cases they have nearly the same effect. Description of the other mechanism will have to wait until a later date; it is believed that the sex-linked and autosomal recessives and many of the dominants have been induced by the Mutator gene here under consideration.

Since tests for allelism were not generally applied, the number of occurrences of a certain mutation can be given only for those loci where identification by phenotype is not likely to lead to confusion with other mutants. The asterisks indicate that the mutation has occurred at least once, but the nature of the mutant does not permit identification by inspection. Thus, dusky and miniature cannot be distinguished phenotypically; although many mutations of this type have appeared, it cannot be stated how many of each. The bar signifies that the mutant has not been observed.

The mutants were given the names of the *D. melanogaster* mutants which they resemble most closely. Some cannot readily be compared. Hairless^{A} is a dominant, lethal to the male. Both prune alleles always show a distinct mottling effect. The deltex alleles are female sterile and semilethal

TABLE 3

X-CHROMOSOME			II CHOROMOSOME		
lozenge (*lz*)	0.0	*	Smoky (*Sm*)	0.0	40
almondex (*amx*)	3.6	*	Thorax (*Tho*)	5.1	2
beaded (*bd*)	31.1	50	Stubble (*Sb*)	6.1	10
Minute[4] (*M*[4])	35.5	*	Hairless[E] (*H*[E])	14.9	*
Hairless[A] (*H*[A])	54.1	*	bithorax (*bx*)	39.2	—
scutellar (*sct*)	61.9	39	glass (*gl*)	42.1	*
yellow (*y*)	61.9	6	cinnabar (*cn*)	54.7	—
prune (pn)		2			
deltex (*dx*)	64.7	3	Minute[8] (*M*[8])		*
Notch (*N*)		24	Minute[14] (*M*[14])		*
white (*w*)	69.6	3	Mutator (*Mu*)	?	
lm$_1$*B*		—			
			III CHROMOSOME		
singed (*sn*)	71.0	*			
vermilion (*v*)	72.8	*			
Filiform (*Ff*)	72.9	*			
lethal plexus (*lp*)	73.1		Abbreviated (*Abb*)	0.0	*
Fused[2] (*Fu*[2])	73.5	*	Lobe (*L*)	29.2	*
dusky (*dy*)	73.6	*	Scute (*Sc*)	50.5	*
miniature (*m*)	73.6	*	Vestigial (*Vg*)	50.5	*
forked (*f*)	74.1	*			
rose (*rs*)	77.2	—	gap (*gp*)		*
tilt (*tt*)	83.7	11	orange (*or*)		*
bubble (*bbl*)	91.6	*	Minute[5] (*M*[5])		*
fused[1] (*fu*[1])	92.3	*	Minute[6] (*M*[6])		*
compressed (*com*)		5	Minute[7] (*M*[7])		*
ascute (*asc*)	98.9	10	Minute[10] (*M*[10])		*
Abnormal wing (*Aw*)	99.8	7	Minute[12] (*M*[12])		*
Curvoid (*Cur*)	106.2	*	Minute[15] (*M*[15])		*
scarlet (*st*)	119.3	*			
lm$_3$*B*		—			
			IV CHROMSOME		
sepia (*se*)	149.0	—			
radius rudimentary (*rr*)	171.4	—			
javelin (*jv*)	176.2	*	Minute[3] (*M*[3])	0.0	*
short (*s*)	181.8	3	Scutellum diminished (*Sd*)	32.9	*
			Hairless[B] (*H*[B])	57.2	*
radius incompletus (*ri*)		*	Curly (*Cy*)	69.7	*
shiny (*shi*)		*	Dachsoid (*Dsd*)		*
tiny bristle (*tb*)		*			
condensed (*con*)		*	net (*net*)		*
roughex (*rux*)		*	dachs (*d*)		*
notchy (*ny*)		*	Large wing cells (*Lwc*)		*
downy (*dow*)		*	Enhancer-glass (*E-gl*)		*
diminutive (*dm*)		*	Minute[1] (*M*[1])		*
small bristle (*sbr*)		*	Minute[2] (*M*[2])		*
ruby (*rb*)		*	Minute[9] (*M*[9])		*
approximated (*app*)		*	Minute[11] (*M*[11])		*
twisted (*tw*)		*	Minute[13] (*M*[13])		*

to the male. Abnormal wing has large wing cells giving the wing a coarse appearance; delta or net type of venation is common; it is usually lethal to the male. In shiny the chitin has a polished appearance. Thorax shows a longitudinal median groove of the thorax as the most striking characteristic. Hairless^{E} rarely survives when homozygous; in the homozygous state it shows extreme aristopedia effects; the heterozygous flies sometimes have the aristae reduced or absent; the legs also are sometimes reduced. Vestigial takes off some bristles along the costa when heterozygous. Scutellum diminished affects the size of the scutellum which may be practically absent in extreme cases. Hairless^{B} resembles heterozygous Hairless^{E}. Dachsoid shortens the legs and roughens the eyes. Large wing cells resembles Abnormal wing in its phenotypic effect. Enhancer-glass allows heterozygous glass to express a rough eye.

In addition to the mutants shown in the maps there has occurred a large number of mutants which were either inviable or sterile, lost or discarded before being mapped, or which have not yet been mapped. The total number of observed mutations is approximately two thousand.

Aside from the evolutionary significance of Mutator genes it is interesting to speculate about their mode of action. From the nature of the mutations which are induced by this mechanism it seems obvious that we are dealing with true point mutations. Mutator genes probably act through a chemical medium. Chemicals are the least satisfactory agents of the more important ones which have been investigated as to their faculty of inducing mutations. It is possible that Mutator genes interfere with the proper reproduction of genes and that by learning the nature of this mechanism we may know more about the chemistry of gene reproduction. Mutator genes rather than producing certain substances might be responsible for the lack of a substance necessary for growth and reproduction of genes in general. There is no good chance at this time to learn much about Mutator genes except the result of their activity, unless the substance involved were in the nature of a hormone; this is unlikely, since intranuclear processes are concerned. It must be left to future research further to illuminate this problem.

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Summary.—1. *Drosophila pseudoöbscura*, race B, has furnished certain stocks with an abnormally high mutation rate.

2. The mutations may occur at any time during the development of the germ line or of the somatic line.

3. The phenomenon is due to a dominant "Mutator" gene which increases the normal spontaneous rate about thirty-four times when heterozygous, and about seventy times when homozygous; this represents a

linear increase in the mutation rate with the dosage of the Mutator gene.

4. The Mutator probably is linked to the second chromosome.

5. A total of approximately two thousand mutations was observed; some of the mutations were located.

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*ON THE PHYSICAL BASIS FOR GENETIC RESISTANCE TO MOUSE TYPHOID, SALMONELLA TYPHIMURIUM**

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With most species wide variations in susceptibility to most diseases exist. Some members of the species die, others show different degrees of morbidity. By suitable genetic techniques it is possible to segregate these different levels of disease resistance into consistently breeding groups, some with nearly complete mortality, some of medium resistance, others with little or no morbidity when exposed to the same dose of the causative organism. Using the mouse, *Mus musculus*, as the host and the typhoid organism, *Salmonella typhimurium*, as the disease causing organism investigations at Iowa State College[1, 2] have segregated a mouse population into six different breeding groups characterized in part by their disease resistance. The first two groups designated as *Ba* and *L* have a mortality of 92 and 87 per cent, respectively, when inoculated with 200,000 bacteria; the *E* and *Z* lines have 47 and 42 per cent mortality; and the *R.I.* and *S* lines have 25 and 14 per cent. Each line is now essentially pure breeding for its resistance to *Salmonella typhimurium*. The purpose of this investigation was to investigate one of the several possible physiological causes for this resistance and thus establish a character basis for the observed genetic resistance. The character chosen for study of this correlation is the cellular constitution of the mouse blood.

For the purposes of this analysis the blood of 45 to 50 mice of each strain was examined for the number of erythrocytes, leucocytes and their proportions.

In our mice the cellular constituents of the blood are shown to vary widely, both in total number and the proportion of the different cell types. This variation seems to be characteristic of blood in general, as we have found similar variations in the published results of other investigators. Two major variables and one minor are known contributors to the variation. The major variables are sex of the mouse and the strain from which it is derived. The minor variable is the short age range over which the mice were tested. Analysis of the effect of these variables proves that sex is relatively unimportant in its influence on either the numbers of erythrocytes or leucocytes or the proportion of the cells composing the leucocytes. In its narrow range, age also is not particularly important as the variation attributable to age is irregular, not showing a consistent trend. Genetic differences which have been segregated into the different strains composing our material account for most of the known variation in the numbers of erythrocytes and leucocytes. The same genetic differences affect the proportions of the cell types composing the leucocytes to only a limited degree.

If we contrast the six different strains we find that the levels of the erythrocytes and leucocytes in the blood are characteristic of the particular strains. In the breeding process the variations of these cells in different mice have been reduced and made characteristically higher or lower in the particular strain. It is found that there is no correlation between the degree of fixation of the erythrocytes and the leucocytes indicating that for the six strains they have been fixed independently of each other. The proportions of the different cells composing the leucocytes do not show the same degree of fixation in this hereditary process as do the cell numbers.

The variation in numbers of leucocytes is highly correlated with the degree of resistance which exists for the particular mouse strain (Fig. 1). Those strains of low resistance have low leucocyte numbers whereas those of high resistance have relatively high leucocyte numbers within the range of the customary variation. In the formation of these strains of mice, those of high and those of low resistance have been established through inbreeding and selection for either high survival value or low survival value to the typhoid disease organism. In the process the leucocyte numbers have also been fixed, the correlation between the leucocyte number for the given strain and its resistance being on the order of 0.9. This fact would be as expected if, basically, there was a direct relation between the number of leucocytes which the organism carried and its potentialities for resisting mouse typhoid.

At the same time that these breeding experiments for disease resistance were going on, inbreeding experiments having no particular relation to disease resistance were also molding other different mouse strains. This inbreeding technique should also lead to fixation of particular types of re-

sistance or leucocyte numbers. Such is found to be the case. One of these strains having a low leucocyte number has high susceptibility to mouse

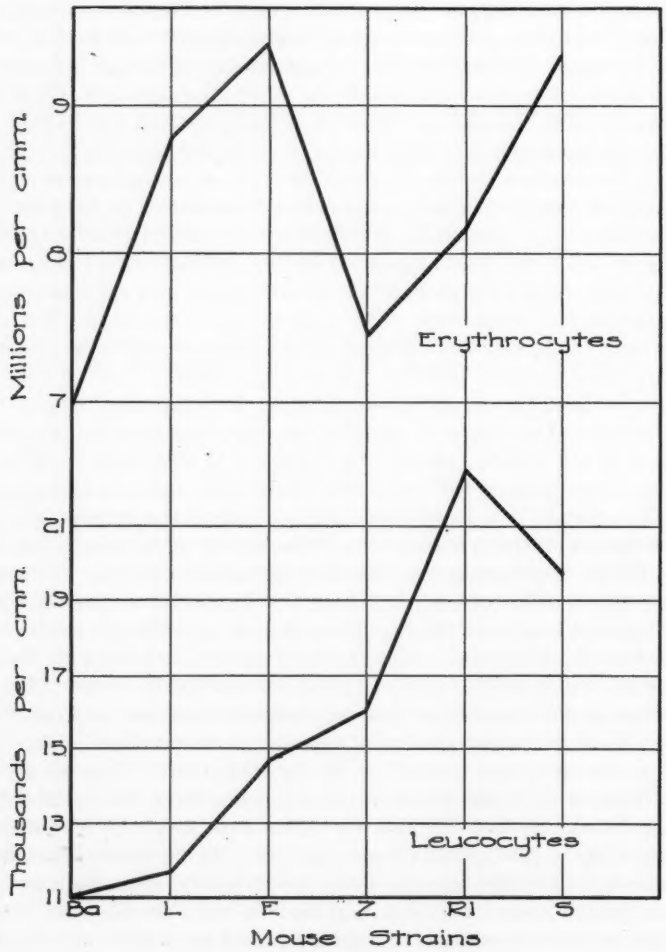

FIGURE 1

Relation of the disease resistance of the different mouse strains to erythrocyte or leucocyte number found in their blood.

typhoid, whereas two of the other strains have intermediate leucocyte number and intermediate susceptibility.

The proportion of the particular types of cells which make up the leucocytes are not fixed in the breeding process. This suggests that the particular cell types are called out by the animal body through different environmental conditions and that a primary cell is capable of developing into any particular type according to these environmental influences rather than through the inheritance control. Such a view would indicate that the important inherited capacity is the production of a few or large number of primary cells that subsequently may develop into leucocytes of the various types according to the environmental need.

The erythrocytes show a similar fixation in their numbers to that of the leucocytes. They do not, however, show any particular correlation to the disease resistance of the given strain. This we might possibly expect in view of the fact that the erythrocytes, so far as mouse blood is concerned, are not known to effect directly the typhoid organism.

In this search for the physical basis of the genetic resistance to mouse typhoid we have studied only two possible types of cells out of many which the body could furnish and be important to the resistance. Both cell types studied have a large variability indicating that there are both hereditary and environmental causes of variation. Analysis shows that our genetic technique would account for about one-fourth of the variations normally present in these mouse blood cells. The other three-fourths of the variation must be due to causes other than those measured in these experiments. The proportions of the different types of cells making up the leucocytes are but little fixed by the genetic techniques used in establishing the different strains.

Our observations have interesting corollaries in the work of Reich and Dunning[3] on the effect of leucocyte level and longevity in rats. Six closely inbred lines of rats were tested for the numbers of white blood cells, the per cent of polymorphs and the duration of life. It was found that the higher the leucocytes the longer the rat lived. It was also shown that the neutrophile polymorph represented a higher proportion of the leucocytes in the longer lived rats than in those with a shorter duration. The correlation is of the order of 0.7. Contrary to our findings a sex effect was noted in the leucocyte count and the duration of life. These sex effects favor larger numbers of leucocytes in the females, the sex with the longer duration of life.

Roberts, Severens and Card[4] present a study of the nature of the hereditary factors for resistance and susceptibility to pullorum in the domestic fowl. In this study they analyze the numbers of erythrocytes, leucocytes, lymphocytes and neutrophiles from the fifteenth day of incubation to a week after the chick hatches. It is in this period that the chicks of susceptible strains are killed by *Salmonella pullorum*. After this period most strains of chickens, both susceptible and resistant, are immune to this dis-

ease. The total number of leucocytes is found to increase in both susceptible and resistant strains from the fifteenth day of incubation through the seventh day after hatching. This increase is accounted for in part by a shift in the types of cells composing the leucocytes. The lymphocytes increase from 5 or 10 per cent up to 55 to 65 per cent of the total leucocytes. The chicks which are genetically resistant to pullorum display this rise in lymphocytes earlier than the chicks which are susceptible. The resistant and susceptible chicks have essentially the same lymphocyte numbers seven days after hatching, the period after which both groups are resistant to this disease.

The importance of the lymphocytes is further brought out by x-ray experiments in which the lymphocytes were reduced in number through irradiating the 6-day-old chicks with x-rays. These x-ray chicks, inoculated with pullorum, had a death rate four times that of the untreated birds. These results would point to the leucocytes as important in the defense mechanism to this disease, especially as no difference in bactericidal power of the serum of the susceptible and resistant groups was observed.

In their study of the correlation between the resistance to rat typhoid and bactericidal power of whole blood, Irwin and Hughes[5] showed that rats which were resistant to the disease had less bacteria in their sodium citrated blood, after inoculation with *Salmonella enteritidis* and incubation at 38° for four hours than rats which were incapable of surviving the disease. As the paper stands it is not possible to decide whether this *in vitro* action is due to the serum or to the presence of leucocytes. We are informed, however, that the serum is probably the responsible agent here.

Rous and Jones[6] have presented a study of the *in vitro* reactions of leucocyte, bacteria or other antigens and immune serum. In this paper they properly emphasize the fact that after one hour's incubation bacteria or red blood cells ingested by leucocytes are protected against the action of immune serum in causing bacteriolysis or hemolysis. They point out the significance of this fact to possible bacterial dissemination within the host should the pathogen be eventually freed from the leucocytes. The protective action against immune sera by the leucocytes is found to be a property of the living organism not of the dead leucocytes. The hypothetical significance of these researches to the possible *in vitro* reactions of the leucocytes to disease organisms is of marked significance to our results. The experimental arrangement is excellent. The experiments present data on pathogen death, or erythrocyte hemolysis, when leucocytes are present in the mixture contrasted with their absence from the mixture. In this light the data show that the leucocytes destroy or immobilize many of the bacteria. This reaction may, of course, be looked upon as an exaggerated form of agglutination since each leucocyte collects a fairly large number of bacteria within it. However, the microscopic examination of ingested

bacteria and erythrocytes would indicate rather that the leucocytes' cytoplasm destroys the inclusions rather than simply agglutinating them.

The general evidence of the foregoing papers indicates that the numbers of leucocytes in general, or numbers of particular kinds of leucocytes, play a pronounced part in the immune phenomena controlled by the genetic constitution of the host.

The original data with its complete analysis will appear shortly.

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BIO-ASSAY ON A GENERAL CURVE

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In the discussion of bio-assay we have used the growth curve[1] where others have used the probability integral. Some of the treatment may be given in terms of a general function F, and for the study of the differences which might be introduced by different laws of biological reaction of various animals to various biologicals such a discussion is not without importance. Let it, then, be supposed that

$$P = 1/2 + 1/2 F[\alpha(x - \gamma)], \quad Q = 1/2 - 1/2 F[\alpha(x - \gamma)]$$

in which α is a coefficient of homogeneity and γ is the 50% point for x so that $F(0) = 0$. For the growth curve and the probability curve we have, respectively,

$$F(z) = \tanh z \qquad \text{and } F(z) = \frac{2}{\sqrt{2\pi}} \int_0^z e^{-z^2/2}\, dz.$$

For each of these curves there is no limit to the values of z, but for some biologicals there might be a minimum dose necessary to produce any effect and a maximum dose which would affect all animals, and in that case z would effectively range only between finite limits. Three hypothetical cases might be

$$F(z) = z, \quad F(z) = \sin z \quad \text{and } F(z) = \frac{z}{\sqrt{1 + z^2}}$$

in the first of which z would range from -1 to $+1$, in the second from $-\pi/2$ to $\pi/2$, and in the third again from $-\infty$ to $+\infty$ but with the order of contact of $F(z)$ with its asymptotes much lower than for the growth curve.

The likelihood is

$$L = \Sigma s_i \log (1 + F_i) + \Sigma(n - s_i) \log (1 - F_i).$$

The equations which determine the parameters α, γ are

$$\frac{\partial L}{\partial \gamma} = -\alpha \sum \frac{2 s_i F_i'}{1 - F_i^2} + n\alpha \sum \frac{F_i'}{1 - F_i} = 0, \qquad (1)$$

$$\frac{\partial L}{\partial \alpha} = \sum \frac{2 s_i (x_i - \gamma) F_i'}{1 - F_i^2} - n \sum \frac{(x_i - \gamma) F_i'}{1 - F_i} = 0. \qquad (2)$$

In general even for the case of three dilutions and $x_i = x - c, x, x + c$, these equations are not generally solvable when F has a specified form.[2]

Differentiating again, we have

$$\frac{\partial^2 L}{\partial \gamma^2} = \alpha^2 \sum \left[\frac{2s_i F_i''}{1 - F_i^2} - \frac{nF_i''}{1 - F_i} + \frac{4s_i F_i'^2 F_i}{(1 - F_i^2)^2} - \frac{nF_i'^2}{(1 - F_i)^2} \right].$$

If we substitute for s_i the value $n[1/2 + 1/2 F_i]$ which it has on the fitted curve, from which any particular set of values s_i is considered to have arisen through sampling errors,

$$\frac{\partial^2 L}{\partial \gamma^2} = -n\alpha^2 \sum \frac{F_i'^2}{1 - F_i^2} \tag{3}$$

and in a similar manner

$$\frac{\partial^2 L}{\partial \gamma \partial \alpha} = n\alpha \sum \frac{(x_i - \gamma) F_i'^2}{1 - F_i^2}, \quad \frac{\partial^2 L}{\partial \alpha^2} = -n \sum \frac{(x_i - \gamma)^2 F_i'^2}{1 - F_i^2}. \tag{4}$$

According to Fisher's formulas[3]

$$\sigma_\gamma^2 = \frac{-\frac{\partial^2 L}{\partial \alpha^2}}{\frac{\partial^2 L}{\partial \alpha^2}\frac{\partial^2 L}{\partial \gamma^2} - \left(\frac{\partial^2 L}{\partial \alpha \partial \gamma}\right)^2} = \frac{\frac{1}{n\alpha^2}\sum \frac{z_i^2 F_i'^2}{1 - F_i^2}}{\sum \frac{z_i^2 F_i'^2}{1 - F_i^2} \sum \frac{F_i'^2}{1 - F_i^2} - \left(\sum \frac{z_i F_i'^2}{1 - F_i^2}\right)^2}, \tag{5}$$

$$\sigma_\alpha^2 = \frac{-\frac{\partial^2 L}{\partial \gamma^2}}{\frac{\partial^2 L}{\partial \alpha^2}\frac{\partial^2 L}{\partial \gamma^2} - \left(\frac{\partial^2 L}{\partial \alpha \partial \gamma}\right)^2} = \frac{\frac{\alpha^2}{n}\sum \frac{F_i'^2}{1 - F_i^2}}{\sum \frac{z_i^2 F_i'^2}{1 - F_i^2} \sum \frac{F_i'^2}{1 - F_i^2} - \left(\sum \frac{z_i F_i'^2}{1 - F_i^2}\right)^2}. \tag{6}$$

It will be observed that in those cases in which the values of x or of $z = \alpha(x - \gamma)$ are so balanced that $\Sigma z F'^2/(1 - F^2) = 0$ or in case α is known the weight of γ is

$$\frac{1}{\sigma_\gamma^2} = n\alpha^2 \sum \frac{F'^2}{1 - F^2}$$

and will be simplest if $F'^2/(1 - F^2)$ is constant. This amounts to having $F(z) = \sin z$ so that there is no effect of the biological up to a certain minimum logarithmic dose $x = \gamma - \pi/2\alpha$ and that all animals are affected by the dose $x = \gamma + \pi/2\alpha$. The solution of the equations (1) and (2) is, however, not simple for $F(z) = \sin z$. On the other hand, the simplest case for the solution of those equations seem to be when $F'/(1 - F^2)$ is con-

stant, i.e., when $F(z) = \tanh z$ and we are working on the growth curve.

If we have determined which type of curve we shall use, the discussion of the precision of determination of γ or of the relative determination of α for different observed values of P_i would be from (4), (5); but if it be desired to compare the precisions that would be figured on the basis of the assumption of two different curve-types as underlying the biological phenomenon we should substitute for α its value in terms of the dilution spread c and the observed values of P_i. To discuss the precision for the general case of any observed values of P_i is impracticable, but for a discussion of the cases in which $F(z)$ is an odd function and there are three values of P_i which are symmetrically distributed as $P_1 = P$, $P_2 = 1/2$, $P_3 = 1 - P$ we may use the expressions

$$\frac{1}{\sigma_\gamma^2} = n\,\frac{\phi^2(1-2P)}{c^2}\left[F'^2_{(0)} + \frac{F'^2_{(z_3)}}{2PQ}\right], \quad \frac{\alpha^2}{\sigma_\alpha^2} = n\,\frac{\phi^2(1-2P)F'^2_{(z_3)}}{2PQ},$$

where $z_3 = \phi(1-2P)$. For the five respective cases of the growth curve, probability integral curve, z, $\sin z$ and $z/\sqrt{1+z^2}$, the results become

$$\frac{c^2}{n\sigma_\gamma^2} = [\tanh^{-1}(1-2P)]^2(1+8PQ),$$

$$V^2(1/2 - P)\left\{0.6366 + 2\frac{\left[\frac{1}{\sqrt{2\pi}}e^{-1/2V^2(1/2-P)}\right]^2}{PQ}\right\},\ (1-2P)^2\left(1+\frac{1}{2PQ}\right),$$

$$3[\sin^{-1}(1-2P)]^2,\ (1-2P)^2\left(\frac{1}{4PQ}+8PQ\right),$$

where ϕ is the function inverse to F and V is the normal variate taken from tables of the probability integral. And, further,

$$\frac{\alpha^2}{n\sigma_\alpha^2} = 8PQ[\tanh^{-1}(1-2P)]^2,\ V^2(1/2-P)2\frac{\left[\frac{1}{\sqrt{2\pi}}e^{-1/2V^2(1/2-P)}\right]^2}{PQ},$$

$$\frac{(1-2P)^2}{2PQ}, \qquad 2[\sin^{-1}(1-2P)]^2, \qquad 8PQ(1-2P)^2.$$

From these expressions we may calculate tables 1 and 2. From table 1 it is clear that in the symmetrical case the values of σ_γ^2 for the growth curve and for the probability integral curve are very nearly the same for any value of P one would be likely to use. It is further clear that for other types of curve assumed as underlying the biological relationship the values of σ_γ may be very different from those found for either of them. It therefore cannot be assumed that σ_γ may not be markedly influenced by the

particular curve-type assumed. What curve-type should be used could only be settled by an extensive series of observations. Moreover, although the analysis has been carried out only for the symmetrical case, yet it seems

TABLE 1

VALUES OF $c^2/(n\sigma_\gamma^2)$ FOR CERTAIN VALUES OF P

	$P=0.40$	$P=0.20$	$P=0.10$	$P=0.05$	$P=0.01$	$P=0$
$F=\tanh z$	0.120	1.10	2.08	2.99	5.70	∞
$F=\frac{2}{\sqrt{2\pi}}\int_0^z e^{-z^2/2}dz$	0.120	1.15	2.17	2.93	4.22	∞
$F=z$	0.123	1.48	4.20	9.34	49.5	∞
$F=\sin z$	0.121	1.24	2.58	3.76	5.63	7.40
$F=z/\sqrt{1+z^2}$	0.118	1.02	2.24	4.57	24.3	∞

reasonable to assume that for cases likely to arise in practice, even though asymmetrical, the growth curve and probability integral curve would yield values of σ_γ more or less alike. The values found for γ itself might also be different in the asymmetrical case, though again it would be reasonable to assume that they would not differ much in ordinary cases.

If it is desired to standardize two biologicals it is of importance to determine whether the two values of α are nearly enough alike so that if the standardization is made at a pair of corresponding points such as the L.D.50 points, it may be regarded as valid for other points. As

$$\sigma\left(\log\frac{\alpha_2}{\alpha_1}\right)=\sqrt{\frac{\sigma_{\alpha_1}^2}{\alpha_1^2}+\frac{\sigma_{\alpha_2}^2}{\alpha_2^2}},$$

we may get some idea of the variation which would be involved in comparing both biologicals on the assumption of different curve-types as underlying the phenomenon, in cases where for each determination the distribution

TABLE 2

VALUES OF $\alpha^2/(n\sigma_\alpha^2)$ FOR CERTAIN VALUES OF P

	$P=0.40$	$P=0.20$	$P=0.10$	$P=0.05$	$P=0.01$	$P=0$
$F=\tanh z$	0.079	0.615	0.870	0.823	0.418	0
$F=\frac{2}{\sqrt{2\pi}}\int_0^z e^{-z^2/2}dz$	0.080	0.694	1.12	1.21	0.777	0
$F=z$	0.083	1.12	3.56	8.53	48.5	∞
$F=\sin z$	0.081	0.828	1.72	2.51	3.75	4.94
$F=z/\sqrt{1+z^2}$	0.077	0.461	0.461	0.308	0.076	0

is symmetrical. From table 2 it will be apparent that the precision of the relative determination of α is by no means so nearly the same for the growth curve and for the probability integral curve as was the case for the determination of γ, and that for other curves it may be very different.

[1] These PROCEEDINGS, 29, 79–85, 114–120 (1943).

[2] If only two dilutions are used and ϕ is the function inverse to F we should have at once

$$\phi(2P_1 - 1) = \alpha(x_1 - \gamma), \quad \phi(2P_2 - 1) = \alpha(x_2 - \gamma),$$

$$\alpha = \frac{\phi(2P_2 - 1) - \phi(2P_1 - 1)}{x_2 - x_1}, \quad \gamma = \frac{x_2 + x_1}{2} - \frac{x_2 - x_1}{2}\,\frac{\phi(2P_2 - 1) + \phi(2P_1 - 1)}{\phi(2P_2 - 1) - \phi(2P_1 - 1)},$$

and the general solution can be obtained whenever the inverse function ϕ may be found. For the respective cases $F = \tanh z$, $F = z$, $F = \sin z$, $F = z/\sqrt{1 + z^2}$ the inverse functions are $\tanh^{-1} F$, F, $\sin^{-1} F$, $F/\sqrt{1 - F^2}$, where F has the value $2P - 1$.

[3] These formulas are those that would result from applying the method of differentiation in the usual manner.

$$\delta\frac{\partial L}{\partial \gamma} = -\sum\left[\frac{2s_iF_i'}{1 - F_i^2} - n\frac{F_i'}{1 - F_i}\right]\delta\alpha + \frac{\partial^2 L}{\partial\alpha\partial\gamma}\delta\alpha + \frac{\partial^2 L}{\partial\gamma^2}\delta\gamma - n\alpha\sum\frac{2F_i'\delta P_i}{1 - F_i^2} = 0.$$

The first term vanishes by virtue of equation (1) which permits the use of equations (3) and (4) for the second derivatives, it being understood that the actual values of s_i are to be considered as having arisen by variations from those on the fitted curve and that δP_i represents variations in the fitted values P_i from a curve with specified parameters α, γ to one with other parameters $\alpha + \delta\alpha$, $\gamma + \delta\gamma$. In this manner we obtain also

$$\delta\frac{\partial L}{\partial \alpha} = \frac{\partial^2 L}{\partial \alpha^2}\delta\alpha + \frac{\partial^2 L}{\partial\alpha\partial\gamma}\delta\gamma + n\sum\frac{2(x_i - \gamma)F_i'\delta P_i}{1 - F_i^2} = 0.$$

If H represents the Hessian determinant,

$$H\delta\gamma = 2n\sum\left[\alpha\frac{\partial^2 L}{\partial\alpha^2} + (x_i - \gamma)\frac{\partial^2 L}{\partial\alpha\partial\gamma}\right]\frac{F_i'\delta P_i}{1 - F_i^2}.$$

As the variations δP_i are considered as independent with standard deviation (squared) of $P_iQ_i/n = {}^1/_4(1 - F_i^2)/n$ we have

$$H^2\sigma_\gamma^2 = n\sum\left[\alpha^2\left(\frac{\partial^2 L}{\partial\alpha^2}\right)^2 + 2\alpha(x_i - \gamma)\frac{\partial^2 L}{\partial\alpha^2}\frac{\partial^2 L}{\partial\alpha\partial\gamma} + (x_i - \gamma)^2\left(\frac{\partial^2 L}{\partial\alpha\partial\gamma}\right)^2\right]\frac{F_i'^2}{1 - F_i^2}.$$

Now substituting from equations (3) and (4) we have

$$H^2\sigma_\gamma^2 = \left[-\left(\frac{\partial^2 L}{\partial\alpha^2}\right)^2\frac{\partial^2 L}{\partial\gamma^2} + 2\frac{\partial^2 L}{\partial\alpha^2}\left(\frac{\partial^2 L}{\partial\alpha\partial\gamma}\right)^2 - \left(\frac{\partial^2 L}{\partial\alpha\partial\gamma}\right)^2\frac{\partial^2 L}{\partial\alpha^2}\right] = -H\frac{\partial^2 L}{\partial\alpha^2}$$

which gives the Fisher formula.

It may be noted that if we desire to obtain a numerical solution of equations (1) and (2) we may proceed by successive approximations. Provided we can find in some way values α_0, γ_0 which nearly satisfy the equations, better values $\alpha_0 + \delta\alpha$, $\gamma_0 + \delta\gamma$ may usually be found from the equations

$$\left.\frac{\partial L}{\partial\gamma}\right|_0 + \left.\frac{\partial^2 L}{\partial\gamma^2}\right|_0\delta\gamma + \left.\frac{\partial^2 L}{\partial\alpha\partial\gamma}\right|_0\delta\alpha = 0, \qquad \left.\frac{\partial L}{\partial\alpha}\right|_0 + \left.\frac{\partial^2 L}{\partial\alpha\partial\gamma}\right|_0\delta\gamma + \left.\frac{\partial^2 L}{\partial\alpha^2}\right|_0\delta\alpha = 0.$$

RELATIONS BETWEEN HOMOLOGY AND HOMOTOPY GROUPS

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1. *The Problem.*—Let P be a locally finite simplicial connected polytope. We denote by $\mathfrak{H}^q(P, G)$ the (discrete) qth homology group of the finite chains of P over a (discrete additive) coefficient group G, by $H_q(P, G)$ the (topologized)[1] qth cohomology group of the infinite cochains of P over a (topologized additive) coefficient group G, by $\pi_q(P)$ the qth homotopy group of P relative to a fixed base point $x_0 \in P$, by $\mathfrak{S}^q(P, G)$ the subgroup of $\mathfrak{H}^q(P, G)$ determined by the cycles of P that can be obtained from cycles on the q-sphere S^q by continuous mappings of S^q into P.

H. Hopf[2] has shown that the fundamental group $\pi_1(P)$ determines the group $\mathfrak{H}^2(P, I)/\mathfrak{S}^2(P, I)$ (I = additive group of all integers) and has exhibited a group construction which leads from π_1 to $\mathfrak{H}^2/\mathfrak{S}^2$. He considers a multiplicative discrete group $\pi = F/R$ represented as a factor group of a free group F by a group of relations R and defines

$$\pi_1^* = R \cap Com\ F/Com_F\ R, \tag{1.1}$$

where *Com* F stand for the commutator group of F, $Com_F\ R$ is the subgroup of F generated by the elements of the form $xrx^{-1}r^{-1}$, $x \in F$, $r \in R$, while "$\cap$" stands for the intersection.

Hopf shows that the group π_1^* does not depend upon the particular representation chosen for π and that if $\pi = \pi_1(P)$ then $\pi_1^* \cong \mathfrak{H}^2(P, I)/\mathfrak{S}^2(P,I)$.

In a more recent paper[3] Hopf has shown that if $\pi_i(P) = 0$ for all $1 < i < n$ then $\pi_1(P)$ determines the group $\mathfrak{H}^n(P, I)/\mathfrak{S}^n(P, I)$. This proof leads to no method for the algebraic determination of $\mathfrak{H}^n/\mathfrak{S}^n$ by means of π_1.

We outline here a new treatment of the problem which leads to generalizations of Hopf's results and gives intrinsic descriptions for all the groups involved. In particular the group π_1^* can be defined intrinsically as the character group[4] of the group of central group extensions[5] of the group X (reals reduced mod 1) by the group π:

$$\pi_1^* = Char\ Extcent\ \{X, \pi\} \tag{1.2}$$

The groups obtained for the higher cases may rightly be regarded as generalizations of the group of group extensions.

The method used also permits us to generalize and simplify the results of Hopf dealing with the influence of $\pi_1(P)$ upon the intersection theory in P.

The results are formulated for a connected polytope P but are valid for any arcwise connected space, provided suitable singular homologies are used.

2. The Complex $K(\pi)$.—Given a discrete group π written multiplicatively we will consider square matrices $\Delta = \| p_{ij} \|$ with p_{ij} in π satisfying the condition

$$p_{ij}p_{jk} = p_{ik}. \tag{2.1}$$

We will denote by $\Delta^{(i)}$ the matrix obtained from Δ by erasing the ith row and the ith column. The rows and columns will always be numbered starting from 0. The matrices Δ with $q + 1$ rows and columns will be taken as the generators of a free abelian group C^q. We define a homomorphism

$$\alpha: C^q \to C^{q-1}$$

by putting

$$\alpha\Delta = \sum_{i=0}^{q} (-1)^i \Delta^{(i)}$$

for each generator Δ of C^q. We verify that $\alpha\alpha = 0$. Consequently if we consider each generator Δ of C^q as a q-cell and α as a boundary operation we obtain a closure finite abstract complex $K(\pi)$. The (discrete) qth homology group obtained by using finite chains of $K(\pi)$ over a (discrete) coefficient group G will be written as $\mathfrak{H}_i^q(\pi, G)$. The (topologized) qth cohomology group obtained using infinite cochains of $K(\pi)$ over a (topologized) coefficient group G will be written as $H_q(\pi, G)$.

3. The Main Theorem.—Let s^q be q-dimensional simplex with vertices $v_0, v_1, \ldots, v_q$ and let $T: s^q \to P$ be a continuous mapping of s^q into the polytope P such that the vertices v_i are all mapped into the point x_0 in P (here x_0 is the base point for the construction of the fundamental group $\pi_1 = \pi_1(P)$). Each edge v_iv_j in s^q will then determine an element p_{ij} in the group π_1 and the matrix $\| p_{ij} \|$ so obtained will satisfy condition (2.1). Hence the mapping T determines a q-cell of the complex $K(\pi_1)$. This observation leads to a chain transformation

$$\tau: P \to K(\pi_1(P)) \tag{3.1}$$

A study of this transformation furnishes the following:

THEOREM 1. *If a connected locally finite polytope P has*

$$\pi_i(P) = 0 \quad \text{for} \quad 1 < i < n, \tag{3.2}$$

the following isomorphisms hold:

$$\mathfrak{H}_i^q(P, G) \cong \mathfrak{H}_i^q(\pi_1(P), G) \quad \text{for} \quad q < n, \tag{3.3}$$

$$H_q(P, G) \cong H_q(\pi_1(P), G) \quad \text{for} \quad q < n, \tag{3.4}$$

$$\mathfrak{H}_i^n(P, G)/\mathfrak{S}^n(P, G) \cong \mathfrak{H}_i^n(\pi_1(P), G). \tag{3.5}$$

4. Computation of the Groups $H_q(\pi, G)$.—We will outline a method for computing the groups $\mathfrak{H}^q(\pi, G)$ and $H_q(\pi, G)$ directly in terms of π and G without using the complex $K(\pi)$. It is sufficient to compute the cohomology groups $H_q(\pi, G)$, since the homology-cohomology duality applied to the complex $K(\pi)$ gives

$$\mathfrak{H}^q(\pi, G) \cong \textit{Char}\, H_q(\pi, \textit{Char}\, G), \quad G \text{ discrete}.$$

Our calculations are based upon a $1 - 1$ correspondence between the matrices Δ with elements in π and the q-tuples $(p_1, \ldots, p_q)$ with $p_i \in \pi$. Given such a q-tuple, the definitions $p_{ii} = 1$, $p_{ij} = p_{i+1} \ldots p_j$ for $i < j$ and $p_{ij} = (p_{ji})^{-1}$ for $j < i$ define a $q + 1$ by $q + 1$ matrix $\Delta = \| p_{ij} \|$ which satisfies (2.1). Conversely, every such matrix Δ can be obtained in this way from exactly one q-tuple. Consequently the group of q-cochains of the complex $K(\pi)$ over the (topologized) group G is nothing but the group $C_q(\pi, G)$ of all functions f of q variables on π to G. The coboundary δf of such a function is a function of $q + 1$ variables defined as follows

$$(\delta f)(p_1, p_2, \ldots, p_{q+1}) = f(p_2, \ldots p_{q+1}) + \sum_{i=1}^{q} (-1)^i f(p_1, \ldots, p_{i-1}, p_i p_{i+1}, p_{i+2}, \ldots, p_{q+1}) + (-1)^{q+1} f(p_1, p_2, \ldots, p_q).$$

The group $Z_q(\pi, G)$ of cocycles consists of the functions f with $\delta f = 0$, while the group $B_q(\pi, G)$ of the cobounding cocycles consists of those functions f of the form $f = \delta g$. The cohomology groups are

$$H_q(\pi, G) = Z_q(\pi, G)/B_q(\pi, G).$$

5. The Cases $q = 1, 2, 3$.—The coboundary of a function of one variable $f: \pi \to G$ is

$$(\delta f)(p_1, p_2) = f(p_2) - f(p_1 p_2) + f(p_1). \tag{5.1}$$

Hence $Z_1(\pi, G)$ is composed of the homomorphisms $f: \pi \to G$. Since $B_1(\pi, G) = 0$ we have

$$H_1(\pi, G) = \textit{Hom}\, \{\pi, G\}.$$

For a function f of two variables the coboundary is

$$(\delta f)(p_1, p_2, p_3) = f(p_2, p_3) - f(p_1 p_2, p_3) + f(p_1, p_2) - f(p_1, p_2). \tag{5.2}$$

The condition that $\delta f = 0$ is exactly the associativity condition for a "central" factor set $f(p_1, p_2)$ of π in G, while the coboundaries δf in (5.1) of functions of one variable are simply the "central" transformation sets. Hence $H_2(\pi, G)$ is the group of factor sets modulo transformation sets, and so is the group of central extensions of G by π,

$$H_2(\pi, G) \cong \textit{Extcent}\, \{G, \pi\}.$$

For $q = 3$ the coboundary δf of f becomes

$$(\delta f)(p_1, p_2, p_3, p_4) = f(p_2, p_3, p_4) - f(p_1p_2, p_3, p_4) + f(p_1, p_2p_3, p_4) - f(p_1, p_2, p_3p_4) + f(p_1, p_2, p_3). \quad (5.3)$$

This operation and the resulting group $H_3(\pi, G)$ have been defined by O. Teichmüller,[6] in a somewhat more general case (corresponding to group extensions which are not necessarily central).

6. *Higher Dimensions.*—A generalization of the main theorem to higher dimensions can be obtained by constructing for each discrete *abelian* group π and each integer $n > 0$ a complex $K(\pi, n)$ whose cells are suitable systems of elements in π with $n + 1$ indices. Denoting the homology and cohomology groups of $K(\pi, n)$ by $\mathfrak{H}_i^q(\pi, n, G)$ and $H_q(\pi, n, G)$ we have

Theorem 2.—Given a connected locally finite polytope P such that

$$\pi_i(P) = 0 \quad \text{for} \quad 0 < i < m \quad \text{and for} \quad m < i < n \quad (6.1)$$

the following isomorphisms hold:

$$\mathfrak{H}_i^q(P, G) \cong \mathfrak{H}_i^q(\pi_m(P), m, G), \quad q < n, \quad (6.2)$$

$$H_q(P, G) \cong H_q(\pi_m(P), m, G), \quad q < n, \quad (6.3)$$

$$\mathfrak{H}_i^n(P, G)/\mathfrak{S}^n(P, G) \cong \mathfrak{H}_i^n(\pi_m(P), m, G). \quad (6.4)$$

[1] The topological groups are understood in a generalized sense, with the Hausdorff separation axiom not postulated.

[2] *Commentarii Math. Helvetici*, **14**, 257–309 (1942).

[3] *Ibid.*, **15**, 27–32 (1942).

[4] The group *Char G* of a given abelian group G is the suitably topologized group of all homomorphisms of the group G into the group X of reals reduced mod 1.

[5] A group E is a central extension of X by H if X is a normal subgroup of E which lies in the center of E and has $E/X = H$. The group of group extensions (including non-central cases) is treated in H. Zassenhaus, *Lehrbuch der Gruppentheorie*, Hamburg Math. Einzelschriften, Leipzig 21, 1937.

[6] O. Teichmüller, "Ueber die sogenannte nichtkommutative Galoische Theorie und die Relation $\xi_{\lambda,\mu\nu}\xi_{\lambda,\mu,\nu}\xi_{\mu,\nu,\pi}{}^{\lambda} = \xi_{\lambda,\mu,\nu\pi}\xi_{\lambda\mu,\nu,\pi}$," *Deutsche Math.*, **5**, 138–149 (1940).

A STRESS FUNCTION FOR THE MEMBRANE THEORY OF SHELLS OF REVOLUTION

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1. The Membrane Theory.—The partial differential equations satisfied by the stresses in a problem concerning the equilibrium of a loaded thin shell or dome of the form of a surface of revolution are very much simplified if it is assumed that the state of stress is independent of any displacements that may occur. The resulting treatment is called "The Membrane Theory." The equations have long been known but seldom solved except in simple cases. The special case when the loads and supports on every meridian of a tank or a dome are the same is solved in the standard engineering texts; the case of a dome loaded or supported non-uniformly has hitherto been solved exactly only for a few surfaces generated by conics, although some approximate methods have been suggested for other cases. If problems of non-uniform support or non-symmetric load are to be treated, it must be within the membrane theory, for the partial differential equations of the ordinary infinitesimal or "bending" theory are so complicated as to render their solution in most cases impracticable.

In this note we produce a simple method of exact solution of the membrane equations for any surface of revolution and for any load representable by a Fourier polynomial; for several families of surfaces of particular interest, we list the solutions explicitly.

2. The Stress Function.—If ϕ is the angle between the normal to the surface and the axis of revolution, θ the angle of rotation measured from some given plane through the axis, R_1 and R_2 the radii of normal curvature, N_ϕ and N_θ the tangential and meridial stresses, $N_{\theta\phi}$ the shear, X, Y, Z, respectively, the parallel, meridial and normal components of the load, then the membrane equations for a shell having the form of a surface of revolution are[1]

$$\left.\begin{aligned} &\frac{N_\phi}{R_1} + \frac{N_\theta}{R_2} = -Z, \\ &\frac{\partial}{\partial\phi}(N_\phi R_2 \sin\phi) - N_\theta R_1 \cos\phi + R_1 \frac{\partial N_{\theta\phi}}{\partial\theta} + Y R_1 R_2 \sin\phi = 0, \\ &R_1 \frac{\partial N_\theta}{\partial\theta} + N_{\theta\phi} R_1 \cos\phi + \frac{\partial}{\partial\phi}(N_{\theta\phi} R_2 \sin\phi) + X R_1 R_2 \sin\phi = 0. \end{aligned}\right\} \quad (1)$$

We shall suppose the stresses and loads represented as Fourier polynomials:

$$\left.\begin{aligned} X &= \sum_{n=0}^{p} X_n(\phi) \sin n\theta, \quad Y = \sum_{n=0}^{p} Y_n(\phi) \cos n\theta, \quad Z = \sum_{n=0}^{p} Z_n(\phi) \cos n\theta, \\ N_\phi &= \sum_{n=0}^{p} N_{\phi n}(\phi) \cos n\theta, \; N_{\theta\phi} = \sum_{n=0}^{p} N_{\theta\phi n}(\phi) \sin n\theta, \; N_\theta = \sum_{n=0}^{p} N_{\theta n}(\phi) \cos n\theta. \end{aligned}\right\} \quad (2)$$

Now let the equation of the generating curve in the $\theta = 0$ plane be $r = f(z)$, where z is measured along the axis of revolution, and let

$$U_n(\phi) \equiv R_2 f(z) N_{\phi n} \sin^2 \phi. \quad (3)$$

If we substitute (2) and (3) in (1), taking advantage of the geometry of the surface, and eliminate $N_{\theta n}$ and $N_{\theta\phi n}$, we are led to the differential equation of the stress function U_n:

$$\frac{d^2U_n}{dz^2} + (n^2 - 1)\frac{f''}{f} U_n = -\frac{d}{dz}[f^2 f' Z_n] + f[1 + (n^2 - 1)(1 + f'^2)]Z_n + f^2 \frac{dY_n}{dz} + 3ff' Y_n + nX_n f\sqrt{1 + f'^2}. \quad (4)$$

When there is no load, the right side vanishes; the equation (4) for this case has been derived previously by Nemenyi from graphostatic considerations.[2] Once the solution of (4) has been obtained for a given surface f, the coefficients of (2) may be found from the formulas

$$\left.\begin{aligned} N_{\phi n} &= \frac{\sqrt{1 + f'^2}}{f^2} U_n, \\ N_{\theta n} &= \frac{f''}{f\sqrt{1 + f'^2}} U_n - f\sqrt{1 + f'^2}\, Z_n, \\ nN_{\theta\phi n} &= -\frac{d}{dz}\left(\frac{U_n}{f}\right) + f(f' Z_n - Y_n). \end{aligned}\right\} \quad (5)$$

Since the method of solution of (4) is well known, we may now consider (1) as solved for all cases when expansions of type (2) are justified. Since, however, to our knowledge there are very few known solutions of (1),[3] it is interesting to write down the explicit solution of (4) for a few surfaces. We shall refer to solutions of (4) with right side zero as "homogeneous solutions"; from them the general solutions of (4) can easily be found.

3. A Few Solutions.—If the equation of the surface f satisfies a differential equation

$$\frac{f''(z)}{f(z)} = A^2 p(z) \tag{6}$$

for some function $p(z)$, then the homogeneous solutions for f can be obtained by putting $-A^2(n^2 - 1)$ for A^2 in the integral of (6), due care being taken to adjust the solutions when this change of constant produces or destroys an integer exponent difference. The indirect method of solving (4) by choosing a function $p(z)$ such that (6) is easily integrable has enabled us to find numerous infinite families of surfaces for which (1) can be solved exactly and in finite form. We list a few of those of possible importance for the engineering applications.

$$\left.\begin{aligned} f &= \sqrt{a-z}\sqrt{b-z}\left[R + S\log\frac{a-z}{b-z}\right], \\ U_n &= \sqrt{a-z}\sqrt{b-z}\left[A_n\left(\frac{b-z}{a-z}\right)^{n/2} + B_n\left(\frac{a-z}{b-z}\right)^{n/2}\right]. \end{aligned}\right\} \tag{7}$$

$$\left.\begin{aligned} f &= R(z+a)^p + S(z+a)^{1-p},\ p \neq 1/2 \\ f &= \sqrt{z+a}[R + S\log(z+a)],\ p = 1/2 \\ U_n &= \sqrt{z+a}[A_n(z+a)^{q_n} + B_n(z+a)^{-q_n}], \end{aligned}\right\} \tag{8}$$

where $q_n^2 \equiv 1/4 - p(p-1)(n^2-1)$.

$$\left.\begin{aligned} f &= \sqrt{z+a}\,B_m(2mA[z+a]^{1/2m}),\ m \neq 0, \\ U_n &= \sqrt{z+a}\,B_m(2miA\sqrt{n^2-1}[z+a]^{1/2m}), \end{aligned}\right\} \tag{9}$$

where $B_m(x)$ is the complete solution of Bessel's equation of order m.

The case $S = 0$, $b = -a$ in (7) gives us, by choice of R, a sphere, a spheroid or a hyperboloid of two sheets; in the first case, the homogeneous solutions reduce to the known solution for the sphere.[4] If we put ia for a, we have a hyperboloid of one sheet. The case $p = 1/2$ of (8) is a paraboloid. The case $m = 1/2$ of (9) gives us a surface generated by the ordinary circular or hyperbolic functions. It is interesting to notice that the solutions for a logarithmic surface are not essentially different from those for an algebraic one; e.g., U_2 for $f = \sqrt{z+a}\log(z+a)$ is the same as U_3 for

$$f = (z+a)^{1/2(1+\sqrt{1/3})}.$$

We have found the solutions for many more surfaces, including those for any surface generated by any solution of the second order equation with two regular singularities, or by any confluent hypergeometric function. There are now enough harmonics found explicitly that it is reasonably easy

to obtain a good approximation to the solution of any soluble statically determinate membrane problem.

4. *Subsequent Results.*—A subsequent investigation by Truesdell alone has produced the following results:

(*a*) A rigorous deduction of an equation similar to (4) whenever the loads can be represented by Fourier series.

(*b*) A complete direct solution of (1) for the case $n = 1$, which is of considerable interest in problems of wind-pressure.

(*c*) The solution of several specimen boundary problems of spheroids, paraboloids and hyperboloids, and a simple method of solution of a dome of any cross-section supported in any manner, and having a lantern bearing a specified load in any manner.

(*d*) A general theorem on the character of the zeros of U_n as related to the zeros of f; a corollary showing that the problem of a closed dome with a zero of order μ at the apex and supported in any non-uniform manner is not soluble within the membrane theory if $\mu \geq 1$, is soluble if $^1/_2 < \mu < 1$ if, and only if, the supports are symmetrically placed and are large enough in number, and is always soluble for two or more symmetrical supports if

$$0 < \mu \leq {}^1/_2.$$

* The work outlined in sections 1 to 3 of this note was done while the authors were fellows at the School of Mathematical Mechanics, Brown University.

[1] See Reissner, H., *Spannungen in Kugelschalen (Kuppeln)*, Festschrift Heinrich Müller Breslau gewidmet, Leipzig, 1912, pp. 181–193, and Flügge, W., *Statik und Dynamik der Schalen*, Berlin 1934, pp. 23–24.

[2] P. Nemenyi, *Beiträge zur Berechnung der Schalen unter unsymmetrischer und unsteliger Belastung*, Bygningsstatiske Meddelelser, 1936.

[3] Flügge, op. cit., Chap. II.

[4] Ibid., p. 39.